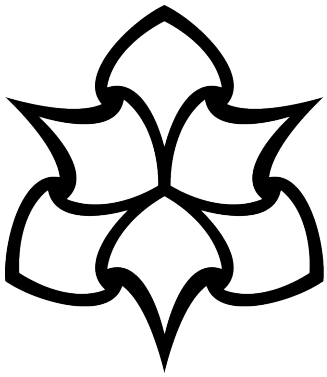

A spatio-temporal and kinematic description of self-selected walking in adults with Achondroplasia

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ABSTRACT

Background: Achondroplasia is characterised by a shorter appendicular limb to torso ratio, compared to age matched individuals of average stature (controls). Despite the well documented shorter leg length of individuals with compared to controls, there are few complete descriptions of gait kinematics reported for the population.
Aim: The aim of this study was to report the spatio-temporal and kinematic characteristics of self-selected walking (SSW) in a group with Achondroplasia (N = 10) and age matched group without Achondroplasia (controls, N = 17).
Method: Whole body 3D analysis of both groups was conducted using a 14 camera VICON system. Spatio-temporal and kinematic variables were determined through a Plug-in-Gait model. SSW was obtained from an average of three trials equating to a total of ~120 m walking.
Results: The group with Achondroplasia were 23 % slower ($P < 0.001$), had a 29 % shorter stride length ($P < 0.001$) and a 13 % higher stride frequency ($P < 0.001$) compared to controls. There were no differences in time normalised temporal measures of left toe off ($P = 0.365$), right heel contact ($P = 0.442$) or the duration of double support ($P = 0.588$) between groups. A number of discrete joint kinematic differences existed between groups, resulting in the group with Achondroplasia having more 'flexed' lower limbs than controls throughout the gait cycle.
Conclusion: Differences in absolute spatio-temporal variables between groups is likely due to the shorter leg length of the group with Achondroplasia, while their more flexed position of the lower limbs may facilitate toe-clearance during the swing phase.

1. Introduction

Achondroplasia is the most prevalent type of skeletal dysplasia and is defined by shorter stature due to disproportionately smaller limb to torso length, compared to age matched able-bodied individuals, hereafter referred to as 'controls' [1–3]. Despite these well documented differences, few investigations have commented on how the shorter lower limb lengths of individuals with Achondroplasia may alter their functional tasks, such as walking. It has recently been shown that the Gait Profile Score (GPS) of adults with Achondroplasia is higher than controls [4]. However, while GPS is useful in describing a global difference in gait between populations, it does not pertain to mechanisms that describe the overall difference in gait. While individuals with Achondroplasia appear to be unaffected by gait limiting pathologies, such as those that are explained by neurological impairment, muscle weakness, amputation or skeletal deformity [5–10], their shorter legs and relatively longer foot (foot-to-leg length ratio) appear to lead to greater knee flexion and ankle dorsiflexion angles during the entire stride compared to controls [4,11]. To date there appear to be four data sets that describe gait and lower limb joint kinematics during self-selected walking (SSW) in groups with Achondroplasia, all of which show subtle differences in kinematic patterns compared to controls [4,11–13], with some suggesting these differences are required to avoid toe contact with the floor during swing [4,11]. None of these studies however, provide a full spatio-temporal or kinematic analysis of gait in any population with Achondroplasia, who have not undergone leg lengthening surgery, to help further explain the differences in kinematics compared to controls. The aim of this study was to therefore describe time normalised kinematic gait patterns of SSW in adults with Achondroplasia who had not undergone leg lengthening surgery and compare these measures to controls. It was hypothesised that there would be kinematic differences between groups for all lower limb

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Table 1
Anthropometric values for the group with Achondroplasia and controls. Values given as mean (min-max, ± SD).

	Achondroplasia		Control
Age (yrs)	22 (18 – 27, ± 3)		22 (19 – 26, ± 2)
Stature (m)	1.38 (1.31 – 1.45, ± 0.05)	*	1.79 (1.55 – 1.88, ± 0.08)
Mass (kg)	61.8 (49.8 – 74.7, ± 8.5)	*	78.3 (61.0 – 77.5, ± 10.7)
Leg Length (m)	0.51 (0.55 – 0.63, ± 0.03)	*	0.86 (0.84 – 1.02, ± 0.05)

* $P \leq 0.001$.

joints, but due to the lack of data describing Achondroplasic gait, these hypotheses are two-tailed.

2. Method

2.1. Participants

After written consent, 10 males with Achondroplasia (Achondroplasic group) and 17 able-bodied males (controls) volunteered to participate in the study (Table 1). All participants reported they were free from lower limb injury and the Achondroplasia group have not undergone leg lengthening surgery. Ethical approval was obtained from the local committee and each participant attended one testing session at a gait laboratory where anthropometric and kinematic assessments at SSW were conducted.

2.2. Kinematic measures

Three-dimensional (3D) motion analysis hardware (VICON, Oxford, UK) was used to determine gait parameters. Anthropometric measures of each participant were taken and entered into the software (Nexus 2.5) to estimate joint centre locations, according to the user manual [14]. Based on Davis' conventional gait model [14], a 39 marker whole-body Plug-in-Gait model was used to obtain centre of mass and lower limb joint kinematics (VICON, Oxford, UK). Fourteen cameras (VICON MX T160, 100 Hz) provided a ~170 m^3 capture area. Participants walked at a self-prescribed speed around the laboratory (~40 m total) and then along ~10 m walkway three times. Each time, a single stride of the left leg, nearest to the centre of the calibration, was used to determine lower limb kinematics of each participant. Gait events were determined using a Bonita camera (720C, 100 Hz). Kinematic data were smoothed using a Butterworth filter with a cut off frequency chosen based on residual analysis of each trial and then time normalised to 101 (i.e. 0–100 %) data points using a publicly available cubic spline interpolation method (Microsoft Excel macro, 2000). Lower limb kinematics were averaged for each participant and SSW was determined by the average speed of the body's centre of mass, calculated using the inertial properties of Dempster [15], for the three strides.

Leg length (m) of all participants was measured as the distance from the anterior iliac spine to the medial malleolus of the ankle while standing. Stride length (m) and stride frequency (Hz) were attained by observing the left and right heel marker in relation to the sagittal plane and floor from the VICON data. Dimensionless values of stride length ($\frac{\text{stride length}}{\text{leg length}}$) and frequency ($\frac{\text{stride frequency}}{\sqrt{9.81 \div \text{leg length}}}$) were also presented for both groups based on the methods of Hof et al. [16]. Temporal events of heel contact and toe off for the left and right sides were calculated as a percentage of total stride time, while the double support phase was calculated as the overlap period of left and right foot floor contact. Based on the recommendations of Benedetti et al. [17], discrete kinematic variables were recorded for the left leg. These were: anterior pelvic tilt (P1), hip flexion (H1), hip internal rotation (H2), knee flexion (K1), ankle plantarflexion (A1) and ankle abduction (A2) at initial heel contact; hip extension (H3), internal hip rotation (H4), knee flexion (K2), knee varus position (K3), plantarflexion (A3) and ankle abduction (A4) at toe off; maximal pelvic tilt (P2), pelvic drop (P3), hip extension (H5), hip abduction (H6), hip adduction (H7), knee flexion (K4), knee varus position (K5), knee valgus position (K6), dorsiflexion (A5) and ankle abduction (A6) during stance phase; maximal anterior pelvic tilt (P4), pelvic drop (P5), hip abduction (H8), hip adduction (H9) and knee flexion (K7) during swing phase, and; maximal internal (P6) and external rotation (P7) of the pelvis for the entire stride. Further to the discrete measures, the mean difference in joint kinematics of the pelvis, hip, knee and ankle between groups were determined over the entire stride for the sagittal plane only.

2.3. Statistical analysis

Statistical analyses were conducted using SPSS (IBM, v24). Data were assumed parametric following normality tests (Shaprio-Wilk) and equal variance tests (Levene's). To account for potential type I errors, a factorial ANOVA was conducted for all spatio-temporal and discrete joint kinematics parameters with only between group comparisons being of interest. Where significant effects were observed in the ANOVA, an independent *t*-tests post hoc with a Bonferroni correction was applied. For all other between group comparisons, an independent samples *t*-test was used. Alpha was set to < 0.05, with all data being presented as mean (min-max, ± SD).

3. Results

3.1. Anthropometric measures

There was no difference in age between groups ($P = 0.487$), but the Achondroplasic group were 23 % shorter in stature ($P < 0.001$), were 19 % lighter ($P < 0.001$) and had a 41 % shorter leg than controls ($P < 0.001$, Table 1).

3.2. Spatio-Temporal

The Achondroplasic group were 23 % slower ($P < 0.001$), had a 29

Table 2
Absolute and normalised spatio and temporal gait parameters of one entire stride during self-selected walking in adults with Achondroplasia adults and age matched controls. Values given as mean (min-max, ± SD).

	Achondroplasia		Control
Gait Speed ($m \cdot s^{-1}$)	1.02 (0.79 – 1.21, ± 0.13)	*	1.33 (1.14 – 1.62, ± 0.14)
Stride Length (m)	0.99 (0.92 – 2.25, ± 0.09)	*	1.40 (1.25 – 1.64, ± 0.10)
Stride Frequency (Hz)	1.07 (0.97 – 1.27, ± 0.08)	*	0.94 (0.83 – 1.06, ± 0.06)
Dimensionless Stride Length	2.19 (2.12 – 2.35, ± 0.08)	*	1.79 (1.43 – 2.06, ± 0.15)
Dimensionless Stride Frequency	0.36 (0.32 – 0.40, ± 0.02)		0.35 (0.26 – 0.40, ± 0.03)
Left Toe off (%)	63 (54 – 66, ± 4)		64 (58 – 79, ± 5)
Right Heel Contact (%)	46 (22 – 53, ± 9)		48 (40 – 52, ± 3)
Double Support (%)	16 (2 – 41, ± 10)		19 (11 – 65, ± 13)

* $P \leq 0.001$.

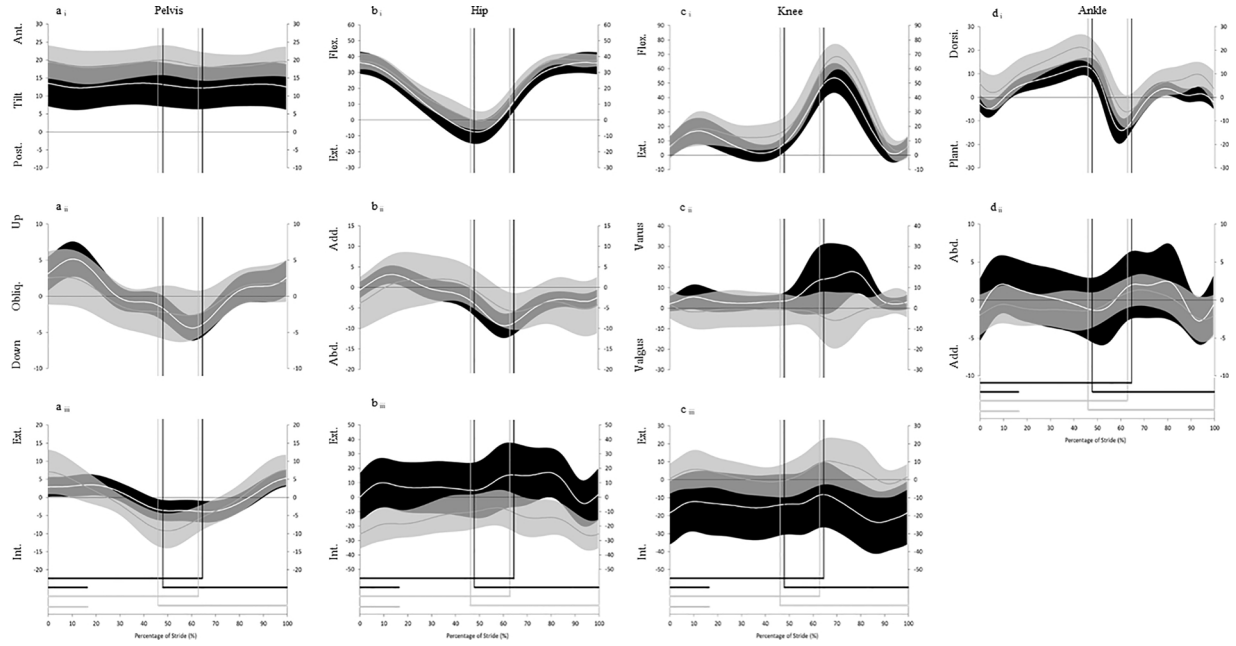

Fig. 1. Joint angles of a) pelvis, b) hip, c) knee, and d) ankle over the same complete stride (%) for the i) sagittal, ii) frontal, and iii) transverse planes (note: ankle eversion is omitted due to inaccuracies of the Plug in Gait model). Grey solid line with grey shading represents the mean (SD) of 3 entire gait cycles for the group with Achondroplasia, while white solid line with black shading represents control mean (SD) of 3 entire gait cycles, respectively. Temporal events are displayed at the bottom of each trio of graphs displaying the: top line) left contact time and bottom line) right contact time. Grey is the group with Achondroplasia and black is control. Vertical lines represent the respective heel contact and toe off points for each leg and are provided for visual interpretation. Pelvis Y axes labels are pelvic tilt (ai) and obliquity (aii) respectively.

% shorter stride length ($P < 0.001$) and a 13 % higher stride frequency than controls ($P < 0.001$, Table 2). When presented as dimensionless values, the group with Achondroplasia had a longer stride than controls ($P < 0.001$) but stride frequency was the same between groups ($P = 0.336$, Table 2). There was no main effect between groups' duration of temporal events when normalised to stride time ($F_{(1,23)} = 0.011$, $P = 0.919$, Table 2 and Fig. 1).

3.3. Discrete variables

There was a significant main effect between groups for the discrete events of the pelvis ($F_{(1,23)} = 4.454$, $P = 0.046$). A greater anterior pelvic tilt at heel contact (P1, $P = 0.020$) and peak tilt during stance (P2, $P = 0.010$) and swing (P3, $P = 0.007$) were observed in the Achondroplasic group compared to controls (Table 3). The Achondroplasic group also exhibited greater maximum external rotation of the pelvis during the stride than controls (P7, $P = 0.005$, Table 3). No differences were observed in any other discrete measure of the pelvis between groups ($P > 0.05$, Table 3). A mean of 5.9° (5.2–7.0, ± 0.5) more anterior tilt during the gait cycle was observed in the Achondroplasic group compared to the control group ($P < 0.001$, Figs. 1ai and 2 b).

There was a significant main effect between groups for the discrete events of the hip ($F_{(1,23)} = 4.357$, $P = 0.048$). The Achondroplasic group exhibited less peak hip extension during the stance phase (H2, $P = 0.023$) and had less internal hip rotation at heel strike (H8, $P < 0.001$) and toe off (H9, $P = 0.005$) compared to controls; there was no difference in any other discrete measure of the hip between groups (Table 3). The Achondroplasic group also used a mean of 4.4° (1.0−7.9, ± 2.0) more hip flexion during the gait cycle than the controls ($P < 0.001$, Figs. 1bi and 2 a).

There was no main effect between groups for the discrete measure of the ankle ($F_{(1,23)} = 0.246$, $P = 0.624$). The Achondroplasic group used 8.1° (0.5−17.3, ± 5.3) more knee flexion during the gait cycle than the controls ($P < 0.001$, Figs. 1ci and 2 a).

There was no main effect between groups for the discrete measure of the ankle ($F_{(1,23)} = 1.998$, $P = 0.171$). The Achondroplasic group did however exhibit 6.5° (2.5−10.9, ± 2.2) more dorsiflexion throughout the gait cycle than controls ($P < 0.001$, Figs. 1di and 2 c).

4. Discussion

This study aimed to describe spatio-temporal and kinematic parameters of the lower limbs during SSW gait in a group of adults with Achondroplasia who had not undergone limb lengthening surgery. The main findings were that adults with Achondroplasia walk slower and have a greater stride frequency than controls and the same group are more 'flexed' at the pelvis, hip, knee and ankle throughout the stride cycle than controls.

The slower SSW of the Achondroplasic group compared to controls was unsurprising given gait speed is determined by stride length, which is in turn determined somewhat by leg length which is short in the Achondroplasic group [4,16]. Despite the difference in SSW speed between groups, no time normalised temporal differences existed, which is consistent with other spatio-temporal observations of gait in shorter versus taller comparisons [18]. Despite the similarities in time normalised temporal events, there were differences in kinematic patterns and discrete events between groups.

One of the main findings in the current study was that the average positions of the pelvis, hip, knee and ankle were more 'flexed' in the Achondroplasic group than controls (Figs. 1 and 2). These data not only confirm the previous sagittal descriptions of groups with Achondroplasia [4,11–13], but also helps explain the larger GPS reported in this population [4]. It has previously been suggested that the gait of individuals with Achondroplasia is more flexed to accommodate toe-clearance during swing phase [11]. One may observe that because the Achondroplasic group had more knee flexion during stance compared to controls (Fig. 1ci), they would require greater knee and ankle flexion of the ipsilateral limb during swing to maintain toe clearance. However, we observed no difference in knee flexion during toe off between

Table 3
Discrete pelvis, hip, knee and ankle kinematics (°) during the average of 3 entire gait cycles of self-selected walking in Achondroplasic adults and age matched controls. Values given as mean (min-max, ± SD).

	Achondroplasia		Control
Anterior pelvic tilt at initial heel contact (P1)	18 (5 – 27, ± 6)	†	13 (1 – 30, ± 6)
Max anterior pelvic tilt during stance phase (P2)	19 (4 – 27, ± 6)	†	14 (2 – 30, ± 6)
Max pelvic rise during stance phase (P3)	4 (-2 – 8, ± 3)		5 (1 – 9, ± 2)
Max anterior pelvic tilt during swing phase (P4)	19 (4 – 26, ± 6)	‡	13 (1 – 30, ± 6)
Max pelvic drop during swing phase (P5)	3 (-2 – 6, ± 2)		3 (0 – 7, ± 2)
Max pelvic internal rotation during the entire stride (P6)	8 (0 – 17, ± 5)		5 (0 – 9, ± 3)
Max pelvic external rotation during the entire stride (P7)	−8 (-20 – 5, ± 6)	‡	−4 (-9 – 3, ± 3)
Hip flexion at initial heel contact (H1)	35 (5 – 46, ± 10)		34 (7 – 51, ± 10)
Hip internal rotation at initial heel contact (H2)	−22 (-38 – 10, ± 14)	*	1 (-31 – 36, ± 16)
Hip extension at toe off (H3)	9 (-8 – 19, ± 7)		8 (-7 – 30, ± 9)
Hip internal rotation at toe off (H4)	−7 (-33 – 13, ± 13)	‡	16 (-33 – 48, ± 21)
Max hip extension during stance phase (H5)	0 (-9 – 9, ± 6)	†	−7 (-18 – 12, ± 8)
Max hip abduction during stance phase (H6)	4 (-8 – 12, ± 5)		3 (0 – 7, ± 2)
Max hip adduction during stance phase (H7)	−5 (-17 – 5, ± 6)		−9 (-16 – 3, ± 4)
Max hip abduction during swing phase (H8)	0 (-13 – 6, ± 5)		−2 (-5 – 1, ± 2)
Max hip adduction during swing phase (H9)	−7 (-20 – 6, ± 7)		−8 (-14 – 3, ± 4)
Knee flexion at initial heel contact (K1)	6 (-5 – 18, ± 7)		6 (-4 – 24, ± 7)
Knee flexion at toe off (K2)	44 (8 – 73, ± 19)		39 (12 – 62, ± 13)
Knee varus angle at toe off (K3)	2 (-9 – 13, ± 6)		2 (-5 – 9, ± 3)
Max knee flexion during stance phase (K4)	44 (8 – 73, ± 19)		40 (13 – 66, ± 14)
Max knee varus angle during stance phase (K5)	5 (0 – 17, ± 5)		15 (-1 – 39, ± 13)
Max knee valgus angle during stance phase (K6)	−10 (-21 – 11, ± 9)		0 (-16 – 7, ± 6)
Max knee flexion during swing phase (K7)	64 (8 – 84, ± 19)		51 (10 – 67, ± 14)
Ankle plantarflexion at initial heel contact (A1)	6 (-2 – 17, ± 6)		−2 (-8 – 6, ± 4)
Ankle abduction at initial heel contact (A2)	9 (-5 – 32, ± 12)		8 (-30 – 40, ± 16)
Ankle plantarflexion at toe off (A3)	−1 (-20 – 8, ± 7)		−9 (-20 – 8, ± 8)
Ankle abduction at toe off (A4)	−6 (-27 – 13, ± 13)		−3 (-37 – 29, ± 18)
Max ankle dorsiflexion during stance phase (A5)	20 (6 – 27, ± 7)		13 (4 – 23, ± 4)
Max ankle abduction during stance phase (A6)	12 (-3 – 32, ± 13)		11 (-30 – 40, ± 18)

† $P \leq 0.05$.
‡ $P \leq 0.01$.
* $P \leq 0.001$.

groups (Table 3, K2). Instead, the Achondroplasic group in this study had a visually larger maximal knee flexion and ankle dorsiflexion during swing phase compared to controls (Figs. 1 and 2) and a possible explanation for this is the relative length of the foot. The average toe-clearance was 18.2 and 23.2 mm for the Achondroplasia and control group, respectively, and both occurred at 81 % of the gait cycle (note: this was measured as the smallest distance in height between the toe marker during swing phase and its lowest position during stance). Were any of the lower limb joints more extended in the Achondroplasic group, toe-clearance would not occur, as demonstrated in Fig. 3. Firstly, changing the knee flexion angle of the Achondroplasic group to that of the controls results in the toe marker being 4.8 mm lower at 81 % of the cycle than its lowest position during stance (Fig. 3a). Secondly, if the Achondroplasic group's ankle plantarflexion was that of the controls, the toe marker is 2.0 mm lower than its lowest position during stance (Fig. 3b). Lastly, changing both the knee flexion and ankle dorsiflexion of the Achondroplasic group results in the toe marker being 24.6 mm lower than its lowest position during stance (Fig. 3c). These examples can be observed somewhat in the study by van der Meulen et al. [13] as their Achondroplasic group had undergone leg lengthening surgery and therefore had a shorter foot (relative to leg length) in comparison to the group included in the current study. A relatively shorter foot reduces the need for flexion at the knee and ankle to avoid toe contact and could explain the visual difference in kinematic patterns between the two studies. Whilst the joint manipulations in Fig. 3 do not show true toe contact with the floor, they do suggest that the positions of the knee and ankle joint in the Achondroplasic group are necessary to maximise toe clearance during swing.

When comparing the presented Achondroplasia kinematic data to other non-leg lengthened groups with Achondroplasia, contradictory data exist. For example, less knee flexion during the entire gait cycle is observed in other Achondroplasic groups compared to the current group [11]. Assuming that marker placements are the same between the current study and Egginton's Achondroplasic group [11], the discrepancy in knee flexion is likely due to a larger hip flexion angle during swing of Egginton's group, thus reducing the need for knee flexion during the swing phase to avoid toe contact with the floor. It is likely therefore, that any individual with Achondroplasia will flex their lower limb joints more than controls during the swing phase to avoid toe-contact with the floor. The amount of flexion though, is dependent on the length of the foot relative to the leg length and is possible there is a greater neural control of the ankle dorsiflexors in the Achondroplasic group to maintain gait [19]. Although, to the author's knowledge, this has not been studied in any Achondroplasic group and is beyond the scope of this study.

Whilst there are subtle differences in sagittal kinematic patterns between the presented and other cohorts with Achondroplasia [4,11–13], there are more substantial differences in the frontal plane kinematics of the knee between the available data sets. In the present study, the Achondroplasic group's knee is in a neutral position during stance phase (Fig. 1cii) whereas children with Achondroplasia exhibit a varus knee position during the stance phase [12,20]. The differences in frontal plane knee kinematics between the groups with Achondroplasia is possibly due to a number of factors. The current group's valgus/varus knee position remains neutral during stance, particularly during the braking phase (Fig. 1cii). This differs to Achondroplasic child who experience a larger valgus/varus range of motion during the stance phase [12]. This may suggest that the compliancy of the knee joint differs between adult and child populations with Achondroplasia, as it does in controls [21]. However, tendon compliancy has only been measured in adults with Achondroplasia [22] and so further work is required to verify this theory. Most likely is that the gait model used is not appropriate to provide accurate frontal knee kinematics for people with Achondroplasia. It has previously been presented that the hip joint

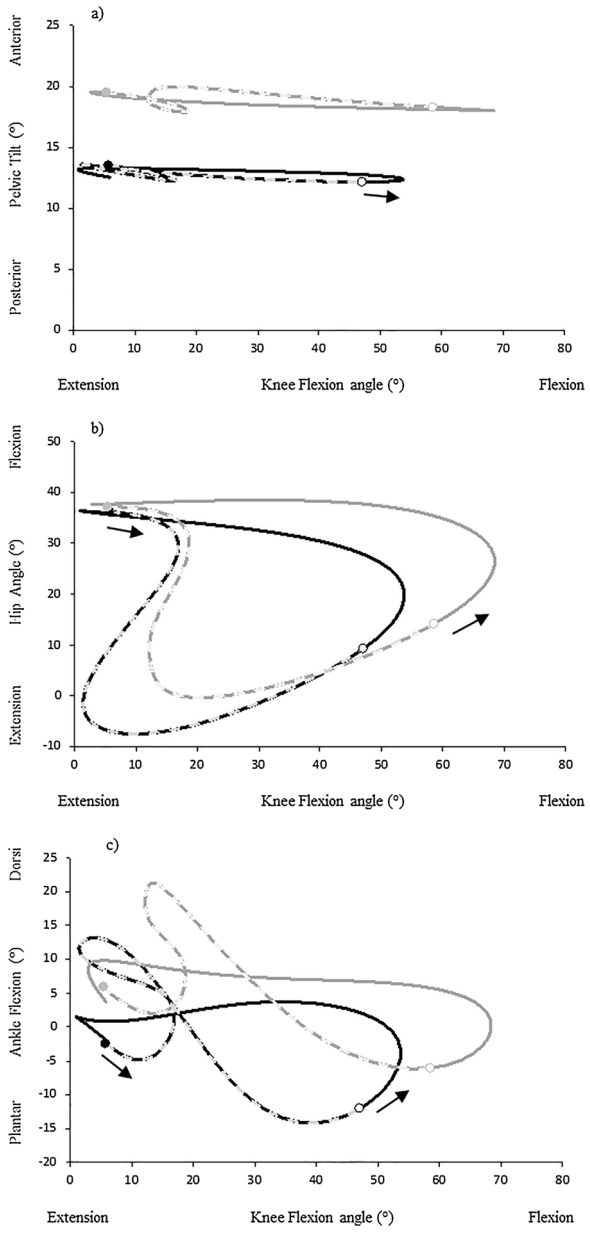

Fig. 2. Angle-angle plots showing the kinematic pattern of a) knee flexion and pelvic tilt, b) knee flexion and hip flexion/extension, and, c) knee flexion and ankle planta/dorsiflexion during an entire stride (%). Grey and black solid lines represent the mean of 3 entire gait cycles for the group with Achondroplasia and controls respectively. Solid dot is initial heel contact, open dot is toe off and dashed line is the period of stance time, respectively. Arrows represent the direction of angular change.

centre (HJC) predictions of people with Achondroplasia differs to controls [4]. These issues likely influence the accurate placement of the thigh marker (relative to the HJC) which then aids in providing internal/external rotation and valgus/varus angles of the knee [23,24]. In the current Achondroplasic group, external hip rotation during stance phase and a knee valgus "wave" during swing phase is observed, suggesting the thigh marker was placed too posteriorly; a knee valgus "wave" during swing is also observed in Achondroplasic children though [12]. The posterior placement of the thigh marker also leads to a more valgus position of the knee during the whole gait cycle [23]. It is possible therefore, that the valgus knee position presented in the current Achondroplasic group are incorrect and that the varus position are under-predicted, but there needs to be a comparison between the gait of adult and child/juvenile populations with Achondroplasia to confirm

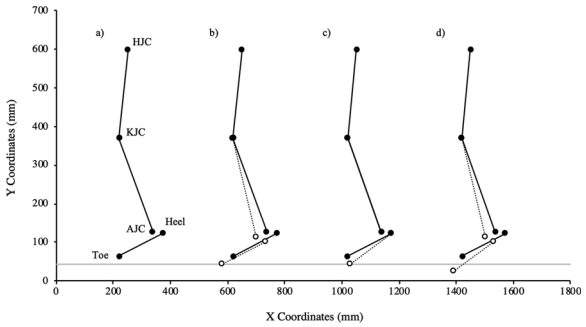

Fig. 3. Sagittal view of the mean hip joint centre (HJC), knee joint centre (KJC), ankle joint centre (AJC), heel and toe for the group with Achondroplasia at 81 % of the gait cycle. Each position is given for a) raw data; b) with the knee angle manipulated to that of controls; c) with the ankle angle manipulated of that of controls; and d) with the knee and ankle angle manipulated to that of controls. In Figures b-d, the projected ankle, heel and toe markers are shown as open white circle with the shank and foot joining each marker as dotted lines; solid black circles and lines are the raw data to show the relative difference in joint position. The solid grey line is position of the toe marker at its lowest point during stance from the raw data.

this. Regardless of the knee valgus/varus position, it is clearly evident that the Achondroplasic group are more flexed during the gait cycle than controls.

4.1. Clinical implications

This study has demonstrated that some gait kinematics differ between individuals with Achondroplasia and controls. From anecdotal evidence and the data presented here and elsewhere [11–13], individuals with Achondroplasia are capable walkers and do not exhibit pain during gait. However, the greater anterior tilt of their pelvis observed in the current study, and reported elsewhere [11,13], is likely to increase shear and compressive forces through the lumbar spine [25]. This could be a precursor for lower back pain, which has been reported in groups with Achondroplasia [11], although further work in this area and in groups with Achondroplasia is required to confirm this link. Were the more anteriorly tilted pelvis to exacerbate lower back pain in individuals with Achondroplasia, strengthening exercises of their weaker hamstrings compared to controls [26] may aid such symptoms. Further work is therefore required to provide prescribed exercise interventions to fully understand the biomechanical mechanisms and physiological responses observed during functional movements in this population. Therapists working with individuals with Achondroplasia should thoroughly scrutinise exercise selection and modality of exercise to ensure that appropriate joints (such as the hip, knee and ankle) go through a range of motion with appropriate load.

5. Conclusion

The current study aimed to present a comprehensive analysis of time normalised lower limb gait kinematics in a homogenous adult population with Achondroplasia who had not undergone limb lengthening surgery. We have demonstrated that this group walk slower and have a higher stride frequency and shorter stride length than controls. Numerous differences in discrete kinematics of the lower limbs exist between the groups, which combine to present a more flexed gait in the Achondroplasic group. The flexed position is likely brought about by a need to avoid toe-contact with the floor.

Declaration of Competing Interest

The authors have no conflict of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.gaitpost.2020.06.030.

References

[1] I.V. Ponseti, Skeletal growth in achondroplasia, J. Bone Jt. Surg. 52 (4) (1970) 701–716.
[2] W.A. Horton, J.I. Rotter, D.L. Rimoin, C.I. Scott, J.G. Hall, Standard growth curves for achondroplasia, J. Pediatr. 93 (3) (1978) 435–438.
[3] J.T. Hecht, O.J. Hood, R.J. Schwartz, J.C. Hennessey, B.A. Bernhardt, W.A. Horton, et al., Obesity in achondroplasia, Am. J. Med. Genet. 31 (3) (1988) 597–602.
[4] D.T. Sims, A. Burden, C. Payton, G.L. Onambélé-Pearson, C.I. Morse, A quantitative description of self-selected walking in adults with Achondroplasia using the gait profile score, Gait Posture 68 (2019) 150–154.
[5] A. van den Hecke, C. Malghem, A. Renders, Cs. Detrembleur, S. Palumbo, T.M. Lejeune, Mechanical work, energetic cost, and gait efficiency in children with cerebral palsy, J. Pediatr. Orthop. Part B 27 (6) (2007) 643–647.
[6] R. Baker, J.L. McGinley, M.H. Schwartz, S. Beynon, A. Rozumalski, H.K. Graham, et al., The gait profile score and movement analysis profile, Gait Posture 30 (3) (2009) 265–269.
[7] S. Beynon, J.L. McGinley, F. Dobson, R. Baker, Correlations of the gait profile score and the movement analysis profile relative to clinical judgments, Gait Posture 32 (1) (2010) 129–132.
[8] R. Baker, J.L. McGinley, M. Schwartz, P. Thomason, J. Rodda, H.K. Graham, The minimal clinically important difference for the Gait Profile Score, Gait Posture 35 (4) (2012) 612–615.
[9] M. Zollinger, F. Degache, G. Currat, L. Pochon, N. Peyrot, C.J. Newman, et al., External mechanical work and pendular energy transduction of overground and treadmill walking in adolescents with unilateral cerebral palsy, Front. Physiol. 7 (2016) 1–10.
[10] R.A. Weinert-Aplin, M. Twiste, H.L. Jarvis, A.N. Bennett, R.J. Baker, Medial-lateral centre of mass displacement and base of support are equally good predictors of metabolic cost in amputee walking, Gait Posture 51 (2017) 41–46.
[11] R. Egginton, C. Newman, M. Walsh, A. Jenkinson, D. Bennett, T. O'Brien, Kinematic characteristics of achondroplasia, Gait Posture 24 (2006) S249–S250.
[12] M. Inan, M. Thacker, C. Church, F. Miller, W.G. Mackenzie, D. Conklin, Dynamic lower extremity alignment in children with achondroplasia, J. Pediatr. Orthop. Part B 26 (4) (2006) 526–529.
[13] J. van der Meulen, W. Dickens, M. Burton, J. Fernandes, O052 Gait characteristics of achondroplasia following lower limb-lengthening, Gait Posture 28 (2008) S36–S37.
[14] R.B. Davis, S. Ounpuu, D. Tyburski, J.R. Gage, A gait analysis data collection and reduction technique, Hum. Mov. Sci. 10 (5) (1991) 575–587.
[15] W.T. Dempster, Space requirements of the seated operator: geometrical, kinematic, and mechanical aspects of the body, with special reference to the limbs (1955).
[16] A.L. Hof, Scaling gait data to body size, Gait Posture 3 (4) (1996) 222–223.
[17] M.G. Benedetti, F. Catani, A. Leardini, E. Pignotti, S. Giannini, Data management in gait analysis for clinical applications, Clin. Biomech. 13 (3) (1998) 204–215.
[18] P.A. Kramer, I. Sarton-Miller, The energetics of human walking: is Froude number (Fr) useful for metabolic comparisons? Gait Posture 27 (2) (2008) 209–215.
[19] T. Killeen, C.S. Easthope, L. Demkó, L. Filli, L. Lőrincz, M. Linnebank, et al., Minimum toe clearance: probing the neural control of locomotion, Sci. Rep. 7 (1) (2017) 1–12.
[20] M.C. Ain, E.D. Shirley, A. Pirouzmanesh, R.L. Skolasky, A.I. Leet, Genu varum in achondroplasia, J. Pediatr. Orthop. Part B 26 (3) (2006) 375–379.
[21] T.D. O'Brien, N.D. Reeves, V. Baltzopoulos, D.A. Jones, C.N. Maganaris, Mechanical properties of the patellar tendon in adults and children, J. Biomech. 43 (6) (2010) 1190–1195.
[22] D.T. Sims, G.L. Onambélé-Pearson, A. Burden, C. Payton, C.I. Morse, Morphological and mechanical properties of the human patella tendon in adult males with Achondroplasia, Front. Physiol. 9 (867) (2018) 1–12.
[23] B.E. Groen, M. Geurts, B. Nienhuis, J. Duysens, Sensitivity of the OLGA and VCM models to erroneous marker placement: effects on 3D-gait kinematics, Gait Posture 35 (3) (2012) 517–521.
[24] R. Baker, Measuring Walking: a Handbook of Clinical Gait Analysis, Mac Keith Press, Hampshire, U.K, 2013.
[25] T.S. Keller, C.J. Colloca, D.E. Harrison, D.D. Harrison, T.J. Janik, Influence of spine morphology on intervertebral disc loads and stresses in asymptomatic adults: implications for the ideal spine, Spine J. 5 (3) (2005) 297–309.
[26] D.T. Sims, G.L. Onambele-Pearson, A. Burden, C. Payton, C.I. Morse, Specific force of the vastus lateralis in adults with Achondroplasia, J. Appl. Physiol. 124 (2018) 696–703.